Amendments to the claims:

This listing of the claims will replace all prior versions, and listings, of claims in the application.

Listing of claims:

Claims 1-16 (cancelled)

Claim 17 (previously presented) A transgenic mouse whose genome comprises a recombinant DNA sequence comprising a nerve tissue-specific promoter operatively linked to a DNA sequence encoding amyloid-beta peptide alcohol dehydrogenase (ABAD), wherein said transgenic mouse overexpresses ABAD in the brain and, relative to a non-transgenic littermate, exhibits at least one phenotype selected from (i) an elevated level of basal ATP and (ii) protection from metabolic or ischemic stress.

Claim 18 (previously presented) The transgenic mouse of claim 17, wherein protection from ischemic stress comprises a decrease in infarct volume or lower neurologic deficit scores in response to ischemic injury relative to a non-transgenic littermate.

Claim 19 (previously presented) The transgenic mouse of claim 17, wherein the promoter is platelet derived growth factor (PDGF)-B-chain promoter.

Claim 20 (previously presented) A method for evaluating in a transgenic mouse the potential therapeutic effect of a compound for treating pathogenesis of Alzheimer's disease in a human, which comprises:

(a) administering the compound to a transgenic mouse whose genome comprises a recombinant DNA sequence comprising a nerve tissue-specific promoter operatively linked to a DNA sequence which encodes amyloid-beta peptide alcohol dehydrogenase (ABAD),

wherein said mouse overexpresses ABAD in the brain and, relative to a non-transgenic littermate, exhibits at least one phenotype selected from the group consisting of (i) an elevated level of basal ATP in cerebral cortex, (ii) a decreased lactate level in cerebral cortex which has been subjected to cerebral ischemia, (iii) more efficient glutamine synthesis, and (iv) a lower beta-hydroxybutyrate level in cerebral cortex which has been subjected to cerebral ischemia; and

(b) determining the therapeutic effect of the compound on the transgenic mouse by monitoring basal synaptic transmission or synaptic plasticity or basal levels of ATP, wherein an increase in basal synaptic transmission or synaptic plasticity or basal levels of ATP indicates that the compound would have a potential therapeutic effect on pathogenesis of Alzheimer's disease in a human.

Claim 21 (previously presented) The method of claim 20, wherein the promoter is platelet derived growth factor (PDGF)-B-chain promoter.

Claim 22 (cancelled)

Claim 23 (currently amended) A transgenic mouse whose genome comprises a recombinant DNA sequence comprising:

(a) a nerve tissue-specific promoter; and

(b) a DNA sequence which encodes amyloid-beta peptide alcohol dehydrogenase (ABAD),

wherein the promoter and the DNA sequence which encodes amyloid-beta peptide alcohol ~~dehyrdogenase~~ dehydrogenase are operatively linked to each other and integrated in the genome of the mouse, and

wherein said mouse overexpresses ABAD in the brain and, relative to a non-transgenic littermate, exhibits at least one phenotype selected from the group consisting of (i) an elevated level of basal ATP in cerebral cortex, (ii) decreased lactate level in cerebral cortex which has been subjected to cerebral ischemia, (iii) more efficient glutamine synthesis, and (iv) lower beta-hydroxybutyrate level in cerebral cortex which has been subjected to cerebral ischemia.

Claim 24 (previously presented) The transgenic mouse of claim 23, wherein the promoter is platelet derived growth factor (PDGF)-B-chain promoter.

Claim 25 (previously presented) The transgenic mouse of claim 23, wherein the DNA sequence which encodes amyloid-beta peptide alcohol dehydrogenase is a human DNA sequence.